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AN OVER VIEW OF THE CLINICAL RELEVANCE OF THE KNOWLEDGE OF THE ORMAL AND ABNORMAL ANATOMY OF THE SELLA TURCICA, USING PLAIN RADIOGRAPHS

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ABSTRACT
Careful examination of the changes within the sella is far more rewarding than measuring the dimensions of the sella. A review was composed via Medline Internet search and literature search. Knowledge of the normal and abnormal anatomy of the sella turcica is significant clinically in the determination of increased intracranial pressure, the determination of direct pressure erosion of the sella from external cases in the immediate vicinity of the sella and the detection of intrasellar expanding lesions. It is concluded that deformity of the sella turcica is often the only clue that abnormality exists within the cranium: hence a familiarity with its anatomy and radiologic appearance is essential.

KEY WORDS: *Sella turcica, knowledge, normal and abnormal anatomy, clinical relevance.*

INTRODUCTION

The sella turcica ('Turkish saddle') is the superior saddle shaped concavity on the intracranial surface of the body of the sphenoid bone (Chummy and Sinnatamby, 2004). It contains the central hypophyseal or pituitary fossa which lodges the hypophyses cerebri or pituitary gland. It has been shown that about 80% of the sella is occupied by the pituitary gland and that the gland may increase in size during pregnancy (DiChiro and Nelson, 1962). It has also been suggested that the sella increases in size with age (Israel, 1970).

The anatomy of the sella turcica is variable in size and shape. It has been classified into three types: round, oval and flat (Jones et al., 2004). It can also be deep or shallow in both children and adults (Isadore, 1976). In profile, the sella at times has a somewhat high concave appearance caused by what appears to be an excavation beneath the anterior clinoids. This is frequently described in children and has no pathological significance. The floor of the sella turcica, which in most cases is concave, may be, flat or even convex (Bruneton et al., 1979).

Careful examination of the changes within the sella is far more rewarding than measuring the dimensions of the sella (Jones et al., 2004).

This paper aims at looking into the relevance of the familiarity with the normal and abnormal anatomy of the sella turcica in clinical practice, using plain radiographs.

Normal anatomy of the sella turcica.

The sella turcica ('Turkish saddle') is the superior saddle shaped concavity on the intracranial surface of the body of the sphenoid bone (Chummy and Sinnatamby, 2004). It contains the central hypophyseal or pituitary fossa which lodges the hypophyses cerebri or pituitary gland. Anteriorly its bony landmarks include the planum sphenoidale, the limbus sphenoidale, the chiasmatic sulcus, and the tuberculum sellae. Anterolateral landmarks include the optic canal, the anterior clinoid processes, and the optic strut, which forms the floor of the optic canal. The floor of the sella is the roof of the sphenoidal air sinus. Posteriorly,

the sella is bounded by the dorsum sellae and the posterior clinoid processes; its lateral margins are the carotid sulci, and its superior boundary is the diaphragma sellae (Newton and Potts, 1971).

The planum sphenoidale: - The planum sphenoidale is a thin well-defined plate of bone that extends from the cribriform plate to the limbus sphenoidale. The central portion, which extends forward and articulates with the cribriform plate, has been termed the ethmoidplate. Its lateral extensions form the roofs of the optic canals and blend with the anterior clinoid prosseses. The planum sphenoidale has a smooth upper surface that may be slightly concave. The width of the planum is determined by the degree of separation between the two orbits (Newton and Potts, 1971).

The limbus sphenoidale:-The limbus sphenoidale marks the posterior boundary of the planum sphenoidale. The appearance of the limbus varies with the shape of the chiasmatic sulcus. When the chiasmatic sulcus is concave, the limbus sphenoidale is prominent. On the other hand, when the chiasmatic sulcus is convex the limbus is barely discernible (Newton and Potts, 1971).

The chiasmatic sulcus: - The chiasmatic sulcus is a depression of variable depth, spanning the distance between the cranial openings of the optic canals. Anteriorly it is bounded by the limbus sphenoidale, and posteriorly the tuberculum sellae. The chiasmatic sulcus is usually horizontal, but it may at times be almost vertical. The width of the chiasmatic sulcus is determined by the width of the planum sphenoidale in an inverse relationship. Thus, a wide planum, leads to a narrow chiasmatic sulcus (Newton and Potts, 1971).

The tuberculum sellae: - The tuberculum sellae is a transverse ridge that forms the posterior margin of the chiasmatic sulcus as well as the anterior margin of the pituitary fossa. It spans the distance between the anterior limits of the carotid sulci. This distance is variable and determines the length of the tuberculum sellae. The prominence of the tuberculum sellae is related to the shape of the chiasmatic sulcus (Newton and Potts, 1971).

The anterior clinoid processes: - The anterior clinoid processes are formed by the medial prolongation of the free posterior margin of the lesser wing of the sphenoid bone. Between the anterior clinoid processes and the tuberculum sellae is a notch which marks the termination of the carotid groove for passage of the internal carotid artery. Thus, the anterior clinoid processes form the anterior and lateral borders of the carotid sulcus. The appearance, shape, and thickness of the anterior clinoid processes vary considerably. They are frequently asymmetric, and occasionally one may be vestigial or absent. The tips of the anterior clinoid processes are situated slightly lateral to the posterior clinoid processes (Newton and Potts, 1971).

The middle clinoid processes: -The middle clinoid processes are in constant elevations on the anterior wall of the pituitary fossa, medial to the carotid artery. At this level the internal carotid arteries pierce the duramater and become intradural. Ossification of the intraclinoid bridge between the anterior and middle clinoid processes leads to the formation of the caroticoclinoid canal (Newton and Potts, 1971). This canal is believed to be a developmental anomaly (Kier, 1968).

The floor of the sella: - For purposes of description, the floor of the sella turcica will include portions of the anterior and the posterior walls of the pituitary fossa. The floor of the sella turcica is therefore considered to extend from the tuberculum to the dorsum sellae. In the sagittal plane the floor usually has a rounded appearance, but its horizontal portion may be relatively straight. In the coronal section the horizontal portion of the floor usually has a slightly convex or flat appearance. Occasionally it has a slight downward concavity. The sellar floor may be asymmetric, sloping downward more on one side than on the other. The horizontal portion of the carotid sulcus forms the lateral margin of the floor. The boundary between the floor and the carotid sulcus is normally rounded (Newton and Potts, 1971).

The carotid sulcus: - The carotid sulcus is a shallow groove along the superolateral aspect of the sphenoid sinus that is formed by the cavernous portion of the internal carotid artery. The depth of the carotid sulcus varies. The sulcus is usually shallow but may have a prominent inferior border (Newton and Potts, 1971).

The dorsum sellae: - The dorsum sellae is the vertical posterior boundary of the sella and is formed by two lateral struts topped by a horizontal strut. It varies greatly in shape and thickness (Newton and Potts, 1971). Mahmoud (1958) found that the thickness of the dorsum ranges from 2 to 7 mm but that only the lateral portions are thick. The central portion is a well-defined hollow between the sturdier lateral pillars and forms a bed for the posterior portion of the pituitary gland. The central portion is often extremely thin. It may even be absent so that a foramen is found within the dorsum sellae (Fry and du Bouley, 1965). The lateral aspect of the dorsum sellae may be grooved by the sixth nerve or by a persistent trigeminal artery. The anterior wall of the dorsum sellae is smooth, whereas the posterior aspect is rough. The roughness of the posterior aspect of the dorsum is due to the dural venous plexus in this location. The posterior clinoid processes are the lateral and superior extensions of the dorsum sellae (Newton and Potts, 1971).

The posterior clinoid processes: - The posterior clinoid processes are the lateral and superior extensions of the dorsum sellae. The tips of the posterior clinoid processes are usually rounded, and they project forward and slightly laterally. Occasionally the tips are pointed and may extend forward to join with the anterior clinoid processes (bridged sella) (Newton and Potts, 1971).

The diaphragma sellae: - The diaphragma sellae is the dural fold formed by the anterior extension of the tentorium. It is attached anteriorly to the tuberculum, posteriorly to the dorsum, and laterally to the interclinoid ligament, which joins the anterior and posterior clinoid processes. The diaphragma sellae has a central opening that transmits the pituitary stalk (Newton and Potts, 1971). Busch (1951) reported that the thickness of the diaphragma, as well as the size of the opening, varies. He examined 788 sellae turcicae and described three main types of diaphragma sellae: (1) diaphragma forms a complete covering with only a small opening for the pituitary stalk (41.9%); (2) the diaphragma is incomplete, with an opening for the stalk less than 3 mm (37.6%); and (3) the diaphragma is represented by only a rim of tissue less than 2 mm in width (20.5%). In a similar study the opening for the pituitary stalk was noted to be greater than 5 mm in 39% of the cases (Bergland, 1968). In sagittal section the diaphragma sellae usually slopes slightly downward into the sella (Newton and Potts, 1971).

Abnormal sella.

Small sella

There is no constant relationship between the size of the sella and the size of the hypophysis. It is very unusual for the sella to be abnormally small. The sella that appears small in the lateral projection may nevertheless be normal because of its greater than average width (Fisher and DiChiro, 1964). A small sella may be found in pituitary hypopituitarism (Riach, 1966), and growth hormone deficiency (Fisher and DiChiro, 1964) if the onset of the disease is before the age of six years. Fisher & DiChiro (1964) found the sella to be small in 56.8% of forty-four subjects with hypopituitarism. Fisher and DiChiro (1964) also found a small sella in 13.5% of subjects with genetic dwarfism. The sella turcica was also reported to be small in a 13½ year old boy with Cushing's syndrome due to an adrenocortical adenoma (Steinbach *et al*., 1963). A small sella was also reported in Sheehan's syndrome (Meador and Worrel, 1959). A small sella in lateral profile may also be found in dystrophia myotica but is not diagnostic of this condition (Caughey, 1952). The sella may be vestigial. This entity has been termed dysplasia of the sella (Lundberg and Gemzell, 1966).

Large sella without local bone destruction

Intrasellar masses may cause enlargement of the sella with preservation of cortical bone. In these instances there is uniform enlargement with a deepening of the floor, and thinning, as well as posterior displacement of the dorsum. Uniform enlargement of the sella is most commonly seen in pituitary tumors, which include chromophobe adenoma, easinophilic adenomas, basophilic adenoma, Nelson's Syndrome, polyadenomatosis, and carcinoma of pituitary. Others are empty sella syndrome, craniopharyngioma, intrasellar aneurysm, hypothyroidism, hypogonadism, neurofibromatosis and oxycephaly (Bergland *et al*., 1968).

Large sella with local bone destruction or erosion

Erosion of undersurface of anterior clinoid processes and chiasmatic sulcus
Erosion of undersurface of anterior clinoid and chiasmatic sulcus can result from glioma of optic chiasm, optic nerve glioma, J sella, optic nerve sheath tumor, pituitary adenoma, aneurysm, dilated third ventricle, craniopharyngioma, frontal lobe tumour, Hunter's syndrome/mucopolysaccharidoses type 11/gargoylism, neurofibromatosis and carotid cavernous fistula (Newton and Potts, 1971).

Erosion of upper surface of anterior clinoid processes
This condition may result from supra-sellar meningioma, increased intracranial pressure due to dilated third ventricle, aneurysm, frontal lobe tumour, suprasellar arachnoid cysts in gargoylism (Newton and Potts, 1971).

Localized erosion of floor
Localized erosion of the sella turcica floor may be due to nasopharyngeal tumor, sphenoid sinus mucocoele, metastases e.g. from carcinoma of the prostate, basal encephalocoele, giant cell tumor, leiomyoma, juvenile nasopharyngeal angiofibroma and postoperative sella (Newton and Potts, 1971).

Erosion of dorsum and posterior clinoid processes
Lateral surface: - This may be affected by parasellar meningioma, neurofibroma, chordoma, aneurysm of internal carotid artery, anomalous vessels, and miscellaneous conditions; e.g. epidermoid and subarachnoid cysts (Newton and Potts, 1971).

Anterior surface of dorsum: - This may be eroded by intrasellar masses, including pituitary tumors (Newton and Potts, 1971).

Upper surface of dorsum and posterior clinoid processes: - These may be eroded by the supra-sellar masses such as craniopharyngioma, optic chiasm glioma, hypothalamic tumor, meningioma, subarachnoid cyst, aneurysm, histiocytosis X, atypical teratoma (ectopic pinealoma), lipoma, dilated third ventricle, frontal glioma and dermoid cyst (Newton and Potts, 1971).

Posterior surface: - The posterior surface of the dorsum sellae may be eroded by posterior fossa tumours (e.g. astrocytoma in children), metastasis (e.g. from colloid carcinoma), arachnoid cyst and basilar artery aneurysm (Newton and Potts, 1971).

Local bony sclerosis and change in texture
This may be brought about by disease conditions like fibrous dysplasia, ossifying fibroma, meningioma, metastatic tumours to the skull, suprasellar tumors (such as craniopharyngioma, atypical teratoma), osteochondroma, osteoma, chondroma of the skull, the clivus, the cerebello pontine angle, and the planum (Minagi and Newton, 1969), soft tissue masses, miscellaneous conditions, such as infections (e.g. tuberculosis, coccidioidomycosis, or pyogenic abscess), trauma (e.g. fracture of the dorsum sellae, floor of the sella and clivus) (Engels, 1961).

Changes in sella in increased intracranial pressure
Erosion of sellar cortex:
Erosion of the cortical bone that lines the pituitary fossa is usually seen earliest at the anterior part of the base of the dorsum. The erosion often spreads to involve most of the sellar floor but occasionally is first recognized more anteriorly (Newton and Potts, 1971). Mahmoud (1958) showed that the process is truly an erosion caused by osteoclasts and that the pits produced in the lamina dura tend to coalesce. In the earlier stages optimal roentgenograms may reveal pinpoint interruptions of the white line of the lamina dura. As erosion progresses, the roentgenographic appearance becomes more obviously abnormal. The outlines of the sella (i.e. lamina dura) are described as rubbed out or osteoporotic (Newton and Potts, 1971).

Erosion of top of dorsum sellae:

Destruction of top of dorsum may include loss of those rough elements of bone that are the site of attachment of the petroclinoid ligaments. As a result of erosion of the posterior clinoid processes, the dorsum in the lateral view may appear as a truncated cone with a flattened top or as a thin spike. In both cases the upper margins are poorly demarcated unless all that remains is a single flake of bone (Newton and Potts, 1971).

Changes in anterior clinoid processes:
The anterior clinoid processes are affected late and in ways different from those of the posterior. In long standing hydrocephalus, the entire base of the skull may be thin. The anterior clinoid processes become thinner and sharper, and in lateral views they may be difficult to recognize. In the posteroanterior inclined projection the anterior clinoid processes and the lesser wings of the sphenoid form frail and indistinct lines (Newton and Potts, 1971).

Enlargement of sella
When the sella enlarges as a result of raised intracranial pressure or hydrocephalus, invariably some erosion or alteration of shape occurs. In raised intracranial pressure the sella may become enlarged in one of three ways: (1) the lamina dura may be destroyed so completely that it no longer has the strength to withstand intracranial pressure as a result the sellar floor herniates downward into the sphenoid sinus; (2) coalescence of erosions in cancellous bone, particularly at the base of the dorsum sellae, may cause the cavity of the sella to extend downward into the clivus, this extension may be appreciated in histologic sections but the cavity does not reach sufficient size to be recognised easily on roentgenograms; (3) enlargement may take place by less dramatic changes in long standing conditions. Erosion is not a feature; but growth under abnormal stimuli causes a peculiar shape of sella. The commonest of these peculiarities is seen sufficiently often to be considered characteristic of chronic obstructive (non-communicating) hydrocephalus. It is not a true enlargement of the sellar cavity itself. The dorsum is short, indeed sometimes extremely so. The longest axis of the sella turcica in the lateral view is more or less in line with clivus. The reason for this appearance is that the anterior sellar wall is elongated upward into the chiasmatic sulcus, which itself is enlarged. Commonly the anterior clinoid processes, presumably because of unusual torsion on their ligaments, are blunt and massive. The upper cavity, limited anteriorly by the chiasmatic sulcus, is occupied by the dilated third ventricle. The cortex is often well formed and without erosions (Newton and Potts, 1971).

Pressure changes of some sort may be noted in about one third of patients with tumors distant from the sella. Of these, 20% do not have papilledema, and amongst them are many who, despite short clinical histories and the absence of other signs of raised intracranial pressure, have longstanding and operable tumors. The earliest detectable abnormality is usually a loss of the lamina dura. Under ideal circumstances this loss is occasionally detected within a few weeks after onset of raised pressure (Tonnis et al., 1954).

Erosion of lamina dura
As an isolated sign, without any other abnormality of the sella turcica, erosion of the lamina dura was found in about 12% of intracranial tumours (Category I erosion) (du Bouley and El Gammal, 1966). In about twice this number of patients with either infra- or supratentorial tumours, erosion of the lamina dura and some additional sellar abnormality were seen. Such erosion occurs in many cases of aqueduct stenosis, but virtually never in communicating hydrocephalus (Newton and Potts, 1971).

Erosion of top and back of dorsum sellae
Tumours of the posterior fossa cause erosion of the top and back of the dorsum sellae (category II erosion) (du Boulay and El Gammal, 1966), about twice as often as do supratentorial tumours. Even so, however, only 10% of posterior fossa tumours are associated with such erosion. In patients with supratentorial tumours, category II change is usually associated with other sellar abnormalities, particularly an extensive erosion of the lamina dura. With hydrocephalus resulting from infratentorial masses, it may be seen alone, however. Still more commonly this type of erosion results from a suprasellar tumour (Newton and Potts, 1971).

Erosion of planum sphenoidale:
After many months of raised intracranial pressure, erosion of the lamina dura may have extended to the planum sphenoidale. The usually crisp white line of that structure blurs or disappears. Nearly always before this disappearance, the top of the dorsum has also been damaged. The combination of erosion of the top of the dorsum sellae, of the lamina dura, and of the planum sphenoidale (category III sellar change) (du Bouley and El Gammal, 1966) is nearly always caused by a slow growing frontal or posterior frontal tumor. Often this tumour is glioma, sometimes a meningioma.

Differentiation between the effects of raised intracranial pressure and the effects of local tumours.
Destruction of the cortical lining of the pituitary fossa may also accompany the growth of tumours, particularly pituitary tumours, within the sella. After thorough examination of the plain roentgenograms of the skull, only about 3% of all pituitary tumours cause confusion regarding diagnosis. The majority of the plain roentgenograms present fairly obvious evidence of the true nature of tumour (Newton and Potts, 1971).

Ballooning of the pituitary fossa is the most common of the additional and specific signs in pituitary adenoma. Sclerosis of bone betrays a meningioma or occasionally a chordoma. Rarely, gliomas of the optic chiasm cause a characteristic excavation in the anterior part of the sellar region. Craniopharyngioma is usually suprasellar and retrosellar. Although a fair number of these tumours protrude into the cavity of the pituitary fossa as well, half of all craniopharyngiomas cause a shortened and extremely flat dorsum. The cortical top of the dorsum in these cases is usually preserved. The abnormality may therefore be distinguished from that caused by frontal tumours and from those producing hydrocephalus (Newton and Potts, 1971).

Differentiation between the changes of pressure in adults and the effect of local tumour and disease is more difficult in the following circumstances: (1) when there is apparently complete destruction of the dorsum sellae; in these situations one should search for displacement of fragments, away from the cavity of the sella (intrasellar tumor), or toward it (hydrocephalus and craniopharyngioma); (2) when, occasionally, erosion of the lamina dura by pituitary adenoma is accompanied by enlargement of the sella, which nevertheless is not characteristically ballooned in shape; (3) rarely, when metastatic carcinoma has spread from the pituitary fossa or the suprasellar region; destruction, or at least an obvious change in texture, extends more deeply into the bone than is the case with raised intracranial pressure (4) when the lesion, usually an aneurysm of the internal carotid, is truly parasellar; in these situations the lateral roentgenogram may suggest at first glance an enlargement and osteoporosis. Careful examination of all views reveals the true state. A faint but sharply outlined dorsum (of which as much as one lateral half may be missing), destruction of one lateral half of the sellar floor, and destruction usually of an anterior clinoid process are all visible (Newton and Potts, 1971).

Changes in sella in childhood.
Similar to adults, the lamina dura in children is eroded in similar situations and after a similar length of time. Changes in sella in childhood may be over looked because diastasis of the sutures attracts the attention of the radiologists. Destruction of the top of the dorsum results particularly from tumours of the posterior fossa that cause hydrocephalus. Rarely destruction may be seen as a result of long-standing frontal and fronto-parietal masses that push the base of the brain against the base of the skull (Newton and Potts, 1971).

In children, however, opportunities for the more chronic sellar changes are fewer. Moreover, slow-growing frontal tumours, which in adults cause many of the most severely damaged sellae, are exceptional before the age of 30. Consequently chronic sellar changes in children tend to be restricted to those suffering from developmental abnormalities, especially aqueduct stenosis. In infancy, changes resulting from communicating hydrocephalus are confined to a simple elongation of the sella, which may accompany a general expansion of the head (Newton and Potts, 1971).

Changes in sella with relief of raised intracranial pressure.
Roentgenograms obtained after excision of cerebral tumors and after operative procedures for the relief of hydrocephalus have been studied. These indicated that osteoclastic resorption ceases rapidly and is replaced by increased new bone formation. Within a few weeks the lamina dura reappeared and some evidence of repair of the dorsum sellae and posterior clinoid processes was seen. The fact that bone (e.g. the posterior clinoid processes) should reappear, must mean that some periosteal or osteoid tissue had not been destroyed entirely. Total destruction and displacement, however, cannot be corrected, so the reformed bone in advanced cases is abnormal in shape. The dorsum may be short, or the cavity of the sella enlarged (Newton and Potts, 1971).

From observation of patients with recurrent symptoms, the susceptibility to erosion of the reformed lamina dura appears to be much less than that of the original bone lining. A second episode of raised intracranial pressure rarely causes erosion (Newton and Potts, 1971).

CONCLUSION

It is concluded that deformity of the sella turcica is often the only clue that abnormality exists within the cranium: hence a familiarity with its anatomy and radiologic appearance is essential.

REFERENCES

Bergland, R.M., Ray B.S., Torack, R.M. 1968. Anatomical variations in the pituitary gland and adjacent structures in 225 human autopsy cases. *J. Neurosurg.*; 28:93-99.

Bruneton, J. N., Drouilard, J.P., Sabatier, J.c., Elie, G.P and Travenir, J.F, 1979. Normal variants of the sella turcica. Comparison of Plain radiographs and tomograms in 200 cases. *Radiology*;131:99-104.

Busch, W. 1951. Die Morphologic der sella turcica und ihre Bexiehungen zur Hypophyse, *Arch. Path. Anat*.; 320: 437-458.

Caughey, J. E. 1952. Bone changes in the skull in dystrophia myotonica, *J. Bone Joint Surgery*.; 34B, 343-359.

Chummy S. and Sinnatamby (2004). (Editors) Last's Anatomy: Regional and Applied; 10th edition. *Churchill Livingstone;* Edinburgh, London,NewYork, Philadelphia, Sydney, Toronto.pp501-504.

DiChiro, G. and Nelson, K. B., 1962. The volume of the sella turcica. *AJR Am. J. Roentgenol*.; 87: 989-1008.

du Boulay, G. H., and El Gammal, T. 1966. Classification, clinical value and mechanism of sella turcica changes in raised intracranial pressure, *Brit. J. Radiol.*; 39: 422-442.

Engels, E. 1958. The roentgen appearance of the carotid sulcus of the sphenoid bone, *Acta Radiol*.; 49: 113-116.

Engels, E. 1961. Basal skull fractures involving the sella turcica. *Clin. Radiol*.; 12: 177-178.

Fisher, R. L. and DiChiro, G. 1964: The small sella turcica. *Amer.Roentgenol.;* 91: 996-1005.

Fry, K. I., and du Boulay, G. H. 1965. Some observations on the sella in old age and arterial hypertension, *Brit. J. Radiol*.; 38: 16-22.

Isadore Meschan M.A. M.D, 1976. An Atlas of Anatomy Basic to Radiology. 1st Edition. *W. B. Saunders Company;* Philadelphia, London, Toronto. pp343-349.

Israel, J. H, 1970. Continuing growth in sella turcica with age, AJR *Am. J. Roentgenol.*; 108: 516-527.

Jones, R.M., Faqir, A., Millet, D.T., Mous, K.F., McHugh, S, 2004. Bridging and Dimensions of Sella Turcica in Subjects Treated by Surgical Orthodontics Means or Orthodontics only. *The Angle Orthodontist*; 75(5): 714-718.

Kier, E.L. 1968. The infantile sella turcica. New roentgenologic and anatomic concepts based on a developmental study of the sphenoid bone. AJR *Am. J. Roentgenol*.; 102:747-767.

Laschi, G. 1931. Un caso di foramen dorsi sellae, *Radiol. Med*.; 18: 707-712.

Lundberg, P. O. and Gemzell, C. 1966. Dysplasia of the sella turcica: Clinical and laboratory investigations in three cases, *Acta Endocrinol*.; 52:478-488.

Mahmoud, M.S. 1958. The sella in health and disease: value of the radiographic study of the sella turcica in morbid anatomical and topographic diagnosis of intracranial tumours, *Brit. J. Radiol*.; Supp. 8, 1-100.

Meador, C. K. and Worrel, J.L. 1959. The sella turcica in post- partum pituitary necrosis (Sheehan's Syndrome). *Ann. Intern. Med*.;65: 252-264.

Minagi, H., and Newton, T. H. 1969. Cartilaginous tumours of the base of the skull, AJR *Am. J. Roentgenol.*; 105: 308-313.

Newton, T.H. and Potts, D.G. 1971. (Editors) Radiology of the skull and brain, The Skull: Volume 1, Book 1; *The CV Mosby Company;* Saint Louis; pp359-405.

Riach, I.C.F. 1966. The pituitary fossa in childhood with particular reference to hypopituitarism, *Brit. J. Radiol*.; 39: 241-243.

Steinbach, H. L., Noetzei, M., and Ozoof, M. B. 1963. Small pituitary fossa in Cushing's syndrome due to adrenal neoplasm, *Neuro Eng. J. Med*.; 269: 1286-1289.

Tonnis, W., Schiefer, W., and Rausch, F. J. 1954. Sella veranderungen bei gesteigerten schadelinnendruck, *Deutsch Z. Nervenheilk.,* 171: 351-369.

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